## De Novo Acute Nonlymphoblastic Leukemia (M5b) Having a Chromosomal 3;21 Translocation With Immunoglobin Heavy-Chain Gene Rearrangement

*To the Editor:* A reciprocal translocation between the long arms of chromosomes 3 and 21 with breakpoints at bands 3q26 and 21q22 [t(3;21)(q26;q22)] has been reported in malignant cells from patients with therapy-related myelodysplastic syndrome (t-MDS), therapy-related acute myeloid leukemia (t-AML), and chronic myelogenous leukemia (CML) [1–3], and it has been reported that the AML1-EVI-1 fusion gene is associated with this translocation [3,4]. However, we report on a rare patient who had de novo acute nonlymphoblastic leukemia with t(3;21) and another interesting feature.

A 67-year-old male was admitted in June 1995 with high fever. Annual medical examinations had revealed no hematologic disorders until then. Physical examination exhibited severe anemia. Hemoglobin was 58 g/l; platelets were 63 $\times$ $10^9$/l; and white blood cell count was 77.2 $\times$ $10^9$/l with 82.5% blasts and 11% monocytes. The following blood chemistry values were recorded: lactate dehydrogenase, 1,230 IU/l; IgG, 3,467 mg/dl; IgA 1,050 mg/dl; IgM, 70 mg/dl; serum lysozyme, 64 μg/ml. M protein, serum IL-1β, and IL-6 were not detected.

Disseminated intravascular coagulation was not revealed. Bone-marrow aspirate was hypercellular, with 82.8% blasts which were negative in peroxidase stain and positive in alpha naphthyl butyrate esterase stain, and with 8% plasma cells which had mild dysplasia (Fig. 1). Surface antigen of blast cells was positive in CD13, CD14, CD33, CD34, CD19, and HLA-DR. Cytogenic studies performed on bone marrow showed 46XY,t(3;21) and Ig heavy-chain rearrangement, but major bcr by RT-PCR was not detected in peripheral blood cells. From the above findings, we diagnosed this case as de novo acute nonlymphoblastic leukemia M5b, with chromosomal abnormality 46XY,t(3;21) and B-lymphoid feature with mild plasma-cell dysplasia. This patient was treated with mitoxantrone (7 mg/m$^2$, days 1–3) and cytosine arabinoside (200 mg/m$^2$, days 1–5). He reached complete remission in October, 1995.

There were two notable points about this case. First, although the 3;21 translocation has been reported in patients with CML, t-AML, t-MDS, and MDS progressing to leukemia, it has very rarely been observed in de novo AML [3]. Secondly, the patient's bone-marrow aspiration revealed mild dysplasia of plasma cells. Akashi et al. [5] reported that simultaneous occurrence of myelomonocytic leukemia and multiple myeloma might originate from common leukemic progenitors. However, since the Ig heavy-chain gene rearrangement disappeared in the remission state of leukemia

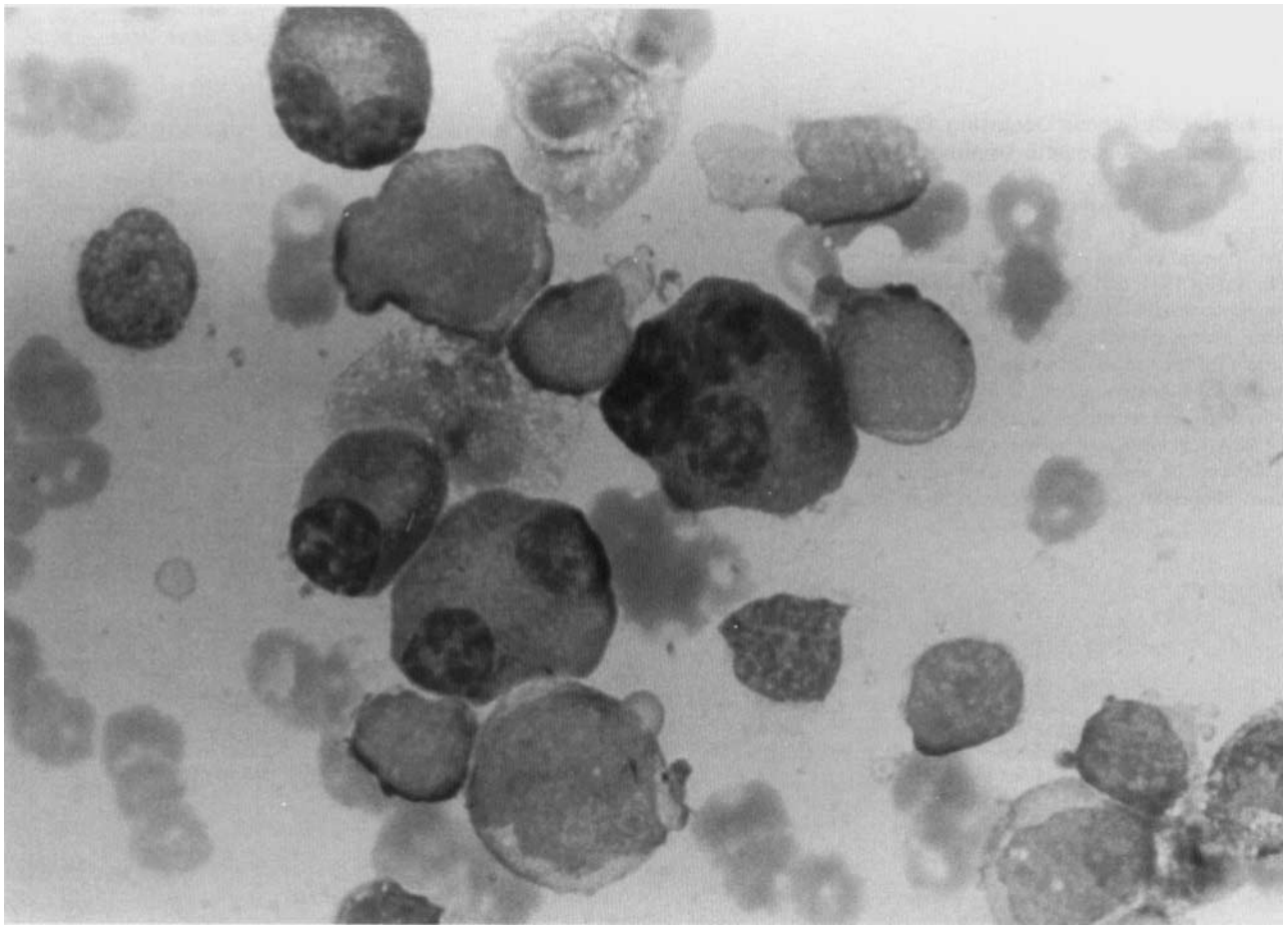

**Fig. 1. Bone-marrow aspirate showed leukemic cells without Auer bodies and plasma cells with mild dysplasia (Giemsa stain, ×1,000). Nuclei of leukemic cells were just like those of monocytes, and had some nucleoli. There were giant plasma cells with multiple nuclei. M protein was never produced in these plasma cells.**

in spite of remaining plasma-cell dysplasia, and since we failed to detect M protein during the patient's hospital course, these plasma cells were not regarded as a neoplasm. It was suggested that leukemic cells with CD19 surface antigen had an Ig heavy-chain gene rearrangement.

In summary, it is suggested that the 3;21 translocation might not rarely be detected also in de novo leukemia and might be associated with B-lymphoid features and plasma cell dysplasia.

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## Hematologic Benefits of 1-Hydroxyvitamin $D_3$ in an Elderly Patient With Chronic Myelodysplastic Syndrome

*To the Editor:* Myelodysplastic syndromes (MDS) represent a heterogeneous group of preleukemic disorders, characterized by advanced age-of-onset and peripheral cytopenias [1]. Among clinical treatments, it has been generally thought that 1,25-dihydroxyvitamin $D_3$ acts to induce differentiation of blast cells in MDS, and that a high dose of 1,25-dihydroxyvitamin $D_3$ produces hypercalcemia [2–4]. We present a patient with MDS who exhibited an elevation of a previously low platelet count and hemoglobin level after the administration of a low dose of 1-hydroxyvitamin $D_3$, such as used in treatment of osteoporosis.

A 70-year-old Japanese woman was admitted to our hospital with purpura on March 3, 1995. Platelet count was $1.0 \times 10^4/\mu l$, hemoglobin level was 8.7 g/dl, and mean corpuscular volume (MCV) was 107.7 fl. The reticulocyte count was $9.2 \times 10^4/\mu l$, and no nucleated red blood cells were found. Examination of a bone-marrow aspirate revealed normocellular marrow with degranulated granulocytes and pseudo-Pelger-Huët anomalies. Blasts constituted 2.4% of all nucleated cells. No chromosomal abnormalities were observed in 20 metaphase cells. Biochemical evaluation showed elevated serum levels of LDH (529 IU/l), hemoglobin-F (1.4%), ferritin (99 ng/ml), and folic acid (11.7 ng/ml), with a normal serum level of vitamin B12 (690 pg/ml). Indirect bilirubin was 0.8 mg/dl. Combs test was negative. The patient was seropositive for the surface antigen of the hepatitis B virus. The level of platelet-associated IgG (PAIgG) was markedly elevated (138.6 $ng/10^7$ cells). Refractory anemia was diagnosed according to the FAB classification. Because of the patient's advanced age and seropositivity for hepatitis B virus, she was administrated only 1 μg of 1-hydroxyvitamin $D_3$ per day. The bicytopenia improved gradually without any side effects: the platelet count rose to $5.4 \times 10^4/\mu l$ and the hemoglobin to 11.1 g/dl, over 10 weeks, and the purpura disappeared. Serum levels of LDH and indirect bilirubin were reduced to 383 IU/l and 0.3 mg/dl, respectively. The PAIgG was reduced to 55.9 $ng/10^7$ cells. On continued treatment for 9 months, the platelet count was maintained at about $5.0 \times 10^4/\mu l$, and the hemoglobin at about 11 g/dl (Fig. 1). A bone-marrow aspirate performed

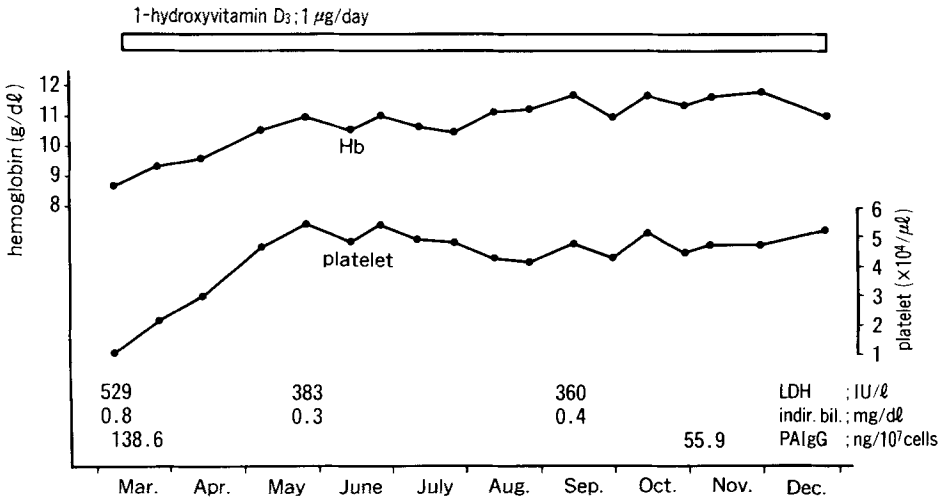


**Fig. 1. Clinical course during treatment with 1-hydroxyvitamin $D_3$. Changes in LDH, indirect bilirubin, and platelet-associated IgG are shown at bottom.**